

Atopic dermatitis (Eczema)

General

Chronic inflammation of epidermis.

Association with allergic rhinitis, asthma.

Effects 1-20% of people worldwide, incidence in US ~10%.

Age of onset typically < 5 years old (85%), of these 40-60% will continue to have eczema into adulthood.

Etiology

Indeterminate etiology; classically thought to be due to heightened immunogenic response to environmental stimuli (similar to asthma, food allergy, allergic rhinitis), however may be due to intrinsic structural abnormalities within the epidermis leading to impaired barrier function.

Risk factors

- higher incidence in Western / industrialized countries, and within these countries incidence increases with socioeconomic status
- childhood eczema often linked to environmental stimuli: too hot or too frequent bathing, low humidity or cold environment
- genetic link may be due to filaggrin gene mutation: creates susceptibility to asthma, allergic rhinitis, atopic dermatitis. *Not* associated with anaphylaxis or food allergy.
- lots of possible risk factors: hard water? maternal asthma? low vitamin D? early antibiotics? psychologic stress?

Presentation

Wide variety of presentations though classically presents as pruritic, erythematous, raised, lichenified, clearly demarcated plaques on flexor surfaces (vs psoriasis, which typically affects extensor surface). Typically spares axilla, genitourinary region.

Diagnosis

Diagnosis is clinical by history and physical examination; no laboratory testing is advised and biopsies are not typically warranted. Food allergy testing not performed in absence of GI symptoms. 0

NICE (National Institute for Health and Clinical Excellence) criteria:

Pruritic skin plus three or more of the following:

- visible dermatitis of flexor surfaces
- history of dermatitis of flexor surfaces
- history of xerosis (dry skin) in past 12 months
- history of asthma or allergic rhinitis
- onset < 2 years old

Differential includes infectious dermatitis (especially arthropod bites, scabies, tinea), contact dermatitis, nummular eczema, immunologic conditions (SLE, hyper-IgE syndrome, dermatitis herpetiformis).

Treatment

Home treatment focuses on emollients, particularly petroleum jelly (Vaseline) as it is unscented and unlikely to trigger immunogenic response. Aqueous creams have a high risk of cutaneous adverse effects. Benefit of bathing unclear as bathing can hydrate the skin but evaporation causes drying of the skin; most dermatologists recommend short duration lukewarm baths or showers and immediate use of emollient afterwards.

Topical corticosteroids are first line medical management. Topical calcineurin inhibitors (e.g. tacrolimus) are second line. Phototherapy is recommended as a complement to either topical corticosteroids or calcineurin inhibitors. Oral histamine blockers given for control of pruritus, do not directly affect lesions. Cyclosporine, azathioprine & oral corticosteroids are almost never warranted except in severe cases.

American Family Physician review article:

<http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list%5Fuids=22962911&>

New England Journal of Medicine review article:

<http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=Abstract&list%5Fuids=18385500&>